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E3      0 --> OLEYLOXYETHYLPHOSPHORYLCHOLINE/CN
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E6      1   OLEYLPHENOL/CN
E7      1   OLEYLPHOSPHORYLETHANOLAMINE/CN
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E15     1   OLEYLTRIMETHYLAMMONIUM BROMIDE/CN
E16     1   OLEYLTRIMETHYLAMMONIUM CHLORIDE/CN
E17     1   OLEYLTRIMETHYLENEDIAMINE/CN
E18     1   OLEYLTRIOCTADECYLAMMONIUM BROMIDE/CN
E19     1   OLF-1/EBF TRANSCRIPTION FACTOR (CAENORHABDITIS ELEGANS STRAIN N2
GENE UNC-3)/CN

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 8-AMINO ACID INSERT ISOFORM)/CN
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 LONG ISOFORM 9L)/CN
 E22 1 OLF-1/EBF-LIKE-2(OS) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
 SHORT ISOFORM 03)/CN
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 E24 1 OLFACTOMEDIN (HUMAN CLONE DE10316701-SEQID-623 GENE OLFM1
 ISOFORM 1)/CN
 E25 1 OLFACTOMEDIN (RANA CATESBEIANA PRECURSOR REDUCED)/CN

=> E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25

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 E2 1 OLEYLOXYETHYL CIDOFOVIR/CN
 E3 0 --> OLEYLOXYETHYLPHOSPHORYLCHOLINE/CN
 E4 1 OLEYLOXYPROPYL-N,N-DIMETHYLAMINE/CN
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 ISOFORM 1)/CN
 E25 1 OLFACTOMEDIN (RANA CATESBEIANA PRECURSOR REDUCED)/CN

=> E "OLEYLOXYETHYL"/CN 25

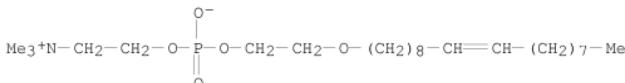
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 E3 0 --> OLEYLOXYETHYL/CN
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E19 1 OLEYLTRIOCTADECYLAMMONIUM BROMIDE/CN
E20 1 OLF-1/EBF TRANSCRIPTION FACTOR (CAENORHABDITIS ELEGANS STRAIN N2
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E21 1 OLF-1/EBF-LIKE-1(8) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
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E22 1 OLF-1/EBF-LIKE-2(9L) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
LONG ISOFORM 9L)/CN
E23 1 OLF-1/EBF-LIKE-2(OS) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
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ISOFORM 1)/CN

=> S 96720-06-8/RN
L1 1 96720-06-8/RN

=> DIS L1 1 SQIDE

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2008 ACS on STN
RN 96720-06-8 REGISTRY
CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
inner salt 4-oxide (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
inner salt, 4-oxide (9CI)
MF C25 H52 N 05 P
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE,
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L2 ANSWER 1 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2004505822 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15474031
TITLE: Interactions of 12-lipoxygenase with phospholipase A2 isoforms following platelet activation through the glycoprotein VI collagen receptor.
AUTHOR: Coffey Marcus J; Coles Barbara; Locke Matthew;
Bermudez-Fajardo Alexandra; Williams P Claire; Jarvis Gavin E; O'donnell Valerie B
CORPORATE SOURCE: Department of Medical Biochemistry and Immunology, Wales College of Medicine, Cardiff University, Heath Park, Cardiff CF14 4XN, UK.. coffeymj@cardiff.ac.uk
SOURCE: FEBS letters, (2004 Oct 8) Vol. 576, No. 1-2, pp. 165-8.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200411
ENTRY DATE: Entered STN: 13 Oct 2004
Last Updated on STN: 19 Dec 2004
Entered Medline: 19 Nov 2004

AB Recent studies implicate the collagen receptor, glycoprotein VI (GPVI) in activation of platelet 12-lipoxygenase (p12-LOX). Herein, we show that GPVI-stimulated 12-hydro(peroxy)eicosatetraenoic acid (H(P)ETE) synthesis is inhibited by palmitoyl trifluoromethyl ketone or oleyloxyethylphosphocholine, but not bromoenol lactone, implicating secretory and cytosolic, but not calcium-independent phospholipase A2 (PLA2) isoforms. Also, following GPVI activation, 12-LOX co-immunoprecipitates with both cytosolic and secretory PLA2 (sPLA2). Finally, venoms containing sPLA2 acutely activate p12-LOX in a dose-dependent manner. This study shows that platelet 12-H(P)ETE generation utilizes arachidonate substrate from both c- and sPLA2 and that 12-LOX functionally associates with both PLA2 isoforms.
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L2 ANSWER 2 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2004455640 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15364285
TITLE: Phospholipase A2 in salivary glands isolated from tobacco hornworms, *Manduca sexta*.
AUTHOR: Tunaz Hasan; Stanley David W
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska-Lincoln, 311 Plant Industry Building, Lincoln, NE 68583-0816, USA.
SOURCE: Comparative biochemistry and physiology. Part B, Biochemistry & molecular biology, (2004 Sep) Vol. 139, No. 1, pp. 27-33.
Journal code: 9516061. ISSN: 1096-4959.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200506
ENTRY DATE: Entered STN: 15 Sep 2004
Last Updated on STN: 1 Jul 2005
Entered Medline: 30 Jun 2005

AB We describe a phospholipase A2 (PLA2) associated with the salivary glands of tobacco hornworms, *Manduca sexta*. This enzyme is able to hydrolyze arachidonic acid from the sn-2 position of PIs. Addition of the calcium chelator, EGTA, or calcium, to the Tris reaction buffer impaired the PLA2 activity, from which we infer the enzyme requires very low concentrations of calcium or perhaps other ions for optimal activity. PLA2 activity was sensitive to protein concentration (highest activity at 25 microg protein per microl), reaction time (optimal at 30 min), buffer pH (optimal at pH 8-10), and reaction temperature (optimal range 18-38 degrees C). The salivary gland PLA2 was sensitive to the site-specific inhibitor, oleyloxyethylphosphorylcholine and stable to freezing at -80 degrees C, but not -20 degrees C. The biological significance of this enzyme may relate to hydrolysis of fatty acid moieties from dietary PIs for absorption by midgut epithelia. This salivary gland enzyme may also be responsible for killing food-borne bacteria.

L2 ANSWER 3 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2003273417 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12798493
TITLE: *lalpha,25(OH)2D3 causes a rapid increase in phosphatidylinositol-specific PLC-beta activity via phospholipase A2-dependent production of lysophospholipid.*
AUTHOR: Schwartz Z; Shaked D; Hardin R R; Gruwell S; Dean D D; Sylvia V L; Boyan B D
CORPORATE SOURCE: Department of Biomedical Engineering, Georgia Institute of Technology, 315 First Drive, Atlanta, GA 30332, USA.
CONTRACT NUMBER: DE-05937 (United States NIDCR)
DE-08603 (United States NIDCR)
SOURCE: Steroids, (2003 May) Vol. 68, No. 5, pp. 423-37.
Journal code: 00404536. ISSN: 0039-128X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 12 Jun 2003
Last Updated on STN: 2 Mar 2004
Entered Medline: 26 Feb 2004

AB *lalpha,25(OH)(2)D(3) activates protein kinase C (PKC) in rat growth plate chondrocytes via mechanisms involving phosphatidylinositol-specific phospholipase C (PI-PLC) and phospholipase A2 (PLA2).* The purpose of this study was to determine if *lalpha,25(OH)(2)D(3)* activates PI-PLC directly or through a PLA(2)-dependent mechanism. We determined which PLC isoforms are present in the growth plate chondrocytes, and determined which isoform(s) of PLC is(are) regulated by *lalpha,25(OH)(2)D(3)*. Inhibitors and activators of PLA(2) were used to assess the inter-relationship between these two phospholipid-signaling pathways. PI-PLC activity in lysates of prehypertrophic and upper hypertrophic zone (growth zone) cells that were incubated with *lalpha,25(OH)(2)D(3)*, was increased within 30s with peak activity at 1-3 min. PI-PLC activity in resting zone cells was unaffected by *lalpha,25(OH)(2)D(3)*. *lbeta,25(OH)(2)D(3), 24R,25(OH)(2)D(3), actinomycin D and cycloheximide* had no effect on PLC in lysates of growth zone cells. Thus, *lalpha,25(OH)(2)D(3)* regulation of PI-PLC enzyme activity is stereospecific, cell maturation-dependent, and nongenomic. PLA(2)-activation (mastoparan or melittin) increased PI-PLC activity to the same extent as *lalpha,25(OH)(2)D(3)*; PLA(2)-inhibition (quinacrine,

oleylxyethylphosphorylcholine (OEPG), or AACOCF(3)) reduced the effect of lalpha,25(OH)(2)D(3). Neither arachidonic acid (AA) nor its metabolites affected PI-PLC. In contrast, lysophosphatidylcholine (LPC) and lysophosphatidylethanolamine (LPE) activated PI-PLC (LPE>LPC). lalpha,25(OH)(2)D(3) stimulated PI-PLC and PKC activities via Gq; GDPbetaS inhibited activity, but pertussis toxin did not. RT-PCR showed that the cells express PLC-beta1, PLC-beta2, PLC-beta3 and PLC-gamma mRNA. Antibodies to PLC-beta1 and PLC-beta3 blocked the lalpha,25(OH)(2)D(3) effect; antibodies to PLC-delta and PLC-gamma did not. Thus, lalpha,25(OH)(2)D(3) regulates PLC-beta through PLA(2)-dependent production of lysophospholipid.

L2 ANSWER 4 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2003052135 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12562098
TITLE: Enzymatic activity and inhibition of the neurotoxic complex
vipoxin from the venom of Vipera ammodytes meridionalis.
AUTHOR: Noetzel Corinna; Chandra Vikas; Perbandt Markus;
Rajashankar Kanagalaghatta; Singh Teji; Aleksiev Boris;
Kalkura Narayana; Genov Nicolay; Betzelt Christian
CORPORATE SOURCE: Institute of Medical Biochemistry and Molecular Biology,
University Hospital Eppendorf c/o DESY, Build. 22a,
Notkestrasse 85, 22603 Hamburg, Germany.
SOURCE: Zeitschrift fur Naturforschung. C, Journal of biosciences,
(2002 Nov-Dec) Vol. 57, No. 11-12, pp. 1078-83.
Journal code: 8912155. ISSN: 0341-0382.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 4 Feb 2003
Last Updated on STN: 16 Apr 2003
Entered Medline: 14 Apr 2003
AB Vipoxin from the venom of Vipera ammodytes meridionalis is an unique neurotoxic complex between a toxic phospholipase A2 and a highly homologous non-toxic protein inhibitor. It is an example of evolution of a catalytic and toxic function into inhibitory and non-toxic one. The activity of the V. ammodytes meridionalis toxin is 1.7 times higher than that of the closely related (92% sequence identity) neurotoxic complex RV4/RV7 from the venom of Vipera russelli formosensis. The enhanced enzymatic activity of vipoxin is attributed to limited structural changes, in particular to the substitutions G54R and Q78K in the PLA2 subunit of the complex and to the T54R substitution in the inhibitor. Oleylxyethylphosphocholine, aristolochic acid and vitamin E suppressed the enzymatic activity of vipoxin and its isolated PLA2 subunit. These compounds influence inflammatory processes in which PLA2 is implicated. The peptide Lys-Ala-Ile-Tyr-Ser, which is an integral part of the PLA2 components of the two neurotoxic complexes from V. ammodytes meridionalis and V. russelli formosensis (sequence 70-74) activated vipoxin increasing its PLA2 activity by 23%. This is in contrast to the inhibitory effect of the respective pentapeptides with 70-74 sequences on other group II PLA2s. Surprisingly, the same peptide inhibited 46% of the V. russelli formosensis PLA2 activity. The limited changes in the structure of the two highly homologous neurotoxins lead to considerable differences in their interaction with native peptides.

L2 ANSWER 5 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2002725402 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12489129
TITLE: Eicosanoids in insect immunity: bacterial infection

stimulates hemocytic phospholipase A2 activity in tobacco hornworms.

AUTHOR: Tunaz Hasan; Park Youngjin; Buyukguzel Kemal; Bedick Jon C; Nor Aliza A R; Stanley David W

CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska, Lincoln 68583-0816, USA.

SOURCE: Archives of insect biochemistry and physiology, (2003 Jan) Vol. 52, No. 1, pp. 1-6.
Journal code: 8501752. ISSN: 0739-4462.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200303

ENTRY DATE: Entered STN: 19 Dec 2002
Last Updated on STN: 4 Mar 2003
Entered Medline: 3 Mar 2003

AB Intracellular phospholipase A(2) (PLA(2)) is responsible for releasing arachidonic acid from cellular phospholipids, and is thought to be the first step in eicosanoid biosynthesis. Intracellular PLA(2)s have been characterized in fat body and hemocytes from tobacco hornworms, Manduca sexta. Here we show that bacterial challenge stimulated increased PLA(2) activity in isolated hemocyte preparations, relative to control hemocyte preparations that were challenged with water. The increased activity was detected as early as 15 s post-challenge and lasted for at least 1 h. The increased activity depended on a minimum bacterial challenge dose, and was inhibited in reactions conducted in the presence of oleyoxyethylphosphorylcholine, a site-specific PLA(2) inhibitor. In independent experiments with serum prepared from whole hemolymph, we found no PLA(2) activity was secreted into serum during the first 24 h following bacterial infection. We infer that a hemocytic intracellular PLA(2) activity is increased immediately an infection is detected. The significance of this enzyme lies in its role in launching the biosynthesis of eicosanoids, which mediate cellular immune reactions to bacterial infection.

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L2 ANSWER 6 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2002147899 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11841807

TITLE: Identification of the phospholipase A(2) isoforms that contribute to arachidonic acid release in hypoxic endothelial cells: limits of phospholipase A(2) inhibitors.

AUTHOR: Michiels Carine; Renard Patricia; Bouaziz Najat; Heck Nathalie; Eliaerts Francois; Ninane Noelle; Quarck Rozenn; Holvoet Paul; Raes Martine

CORPORATE SOURCE: Laboratoire de Biochimie et Biologie Cellulaire, Facultes Universitaires Notre Dame de la Paix, 61 rue de Bruxelles, 5000, Namur, Belgium.. carine.michiels@fundp.ac.be

SOURCE: Biochemical pharmacology, (2002 Jan 15) Vol. 63, No. 2, pp. 321-32.
Journal code: 0101032. ISSN: 0006-2952.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200203

ENTRY DATE: Entered STN: 8 Mar 2002
Last Updated on STN: 3 Apr 2002
Entered Medline: 27 Mar 2002

AB Changes in endothelium functions during ischemia are thought to be of importance in numerous pathological conditions, with, for instance, an increase in the release of inflammatory mediators like prostaglandins. Here, we showed that hypoxia increases phospholipase A(2) (PLA(2)) activity in human umbilical vein endothelial cells. Both basal PLA(2) activity and PG synthesis are sensitive to BEL and AACOCF3, respectively, inhibitors of calcium-independent PLA(2) (iPLA(2)) and cytosolic PLA(2) (cPLA(2)), while OPC, an inhibitor of soluble PLA(2) (sPLA(2)) only inhibited the hypoxia-induced AA release and PGF(2alpha) synthesis. Hypoxia does not alter expression of iPLA(2), sPLA(2) and cPLA(2) and cycloheximide did not inhibit PLA(2) activation, indicating that hypoxia-induced increase in PLA(2) activity is due to activation rather than induction. However, mRNA levels for sPLA(2) displayed a 2-fold increase after 2 hr incubation under hypoxia. BAPTA, an intracellular calcium chelator, partially inhibited the AA release in normoxia and in hypoxia. Direct assays of specific PLA(2) activity showed an increase in sPLA(2) activity but not in cPLA(2) activity after 2hr hypoxia. Taken together, these results indicate that the hypoxia-induced increase in PLA(2) activity is mostly due to the activation of sPLA(2).

L2 ANSWER 7 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2001301904 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11226404
TITLE: The involvement of phospholipase A(2) in ethanol-induced gastric muscle contraction.
AUTHOR: Sim S S; Choi J C; Min D S; Rhie D J; Yoon S H; Hahn S J; Kim C J; Kim M S; Jo Y H
CORPORATE SOURCE: Department of Pathophysiology, College of Pharmacy, Chung-Ang University, 221 Huksuk-dong, Dongjak-gu, Seoul 156-756, South Korea.
SOURCE: European journal of pharmacology, (2001 Feb 16) Vol. 413, No. 2-3, pp. 281-5.
JOURNAL code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 4 Jun 2001
Last Updated on STN: 4 Jun 2001
Entered Medline: 31 May 2001

AB To understand the underlying mechanism of ethanol in tonic contraction, the effect of ethanol on phospholipase A(2) and phospholipase C activities and the effects of phospholipase inhibitors on ethanol-induced contraction of cat gastric smooth muscle were tested. Circular muscle strips (2.0 x 0.2 cm) obtained from the fundus of cat stomach were used to measure isometric contraction. Ethanol elicited tonic contraction and activated phospholipase A(2) activity in a dose-dependent manner. Phospholipase A(2) inhibitors, manaoide (0.1--10 microM) and oleyloxyethyl phosphorylcholine (1--10 microM), significantly inhibited ethanol-induced contraction. Furthermore, 342 mM ethanol-induced contraction was significantly inhibited by cyclooxygenase inhibitors, ibuprofen (10--100 microM) and indomethacin (10--100 microM), but not by lipoxygenase inhibitors. On the other hand, phospholipase C inhibitors had no effect on ethanol-induced contraction, indicating that phospholipase C is not involved in ethanol-induced contraction. It is suggested from the above results that ethanol-induced contraction in cat gastric smooth muscle is, in part, mediated by phospholipase A(2) and cyclooxygenase pathways.

L2 ANSWER 8 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2001121483 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10991918
TITLE: Investigation into the involvement of phospholipases A(2) and MAP kinases in modulation of AA release and cell growth in A549 cells.
AUTHOR: Choudhury Q G; McKay D T; Flower R J; Croxtall J D
CORPORATE SOURCE: Department of Biochemical Pharmacology, The William Harvey Research Institute, St. Bartholomew's and the Royal London School of Medicine and Dentistry (Queen Mary and Westfield College), Charterhouse Square, London EC1M 6BQ.
SOURCE: British journal of pharmacology, (2000 Sep) Vol. 131, No. 2, pp. 255-65.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 22 Feb 2001

AB 1. We have investigated the contribution of specific PLA(2)s to eicosanoid release from A549 cells by using specific inhibitors of secretory PLA(2) (ONO-RS-82 and oleyloxyethylphosphocholine), cytosolic PLA(2) (AACOCF(3) and MAFF) and calcium-independent PLA(2) (HELSS, MAFF and PACOCF(3)). Similarly, by using specific inhibitors of p38 MAPK (SB 203580), ERK1/2 MAPK (Apigenin) and MEK1/2 (PD 98059) we have further evaluated potential pathways of AA release in this cell line. 2. ONO-RS-82 and oleyloxyethylphosphocholine had no significant effect on EGF or IL-1beta stimulated (3)H-AA or PGE(2) release or cell proliferation. AACOCF(3), HELSS, MAFF and PACOCF(3) significantly inhibited both EGF and IL-1beta stimulated (3)H-AA and PGE(2) release as well as cell proliferation. Apigenin and PD 98059 significantly inhibited both EGF and IL-1beta stimulated (3)H-AA and PGE(2) release and cell proliferation whereas, SB 203580 had no significant effect on EGF or IL-1beta stimulated (3)H-AA release, or cell proliferation but significantly suppressed EGF or IL-1beta stimulated PGE(2) release. 3. These results confirm that the liberation of AA release, generation of PGE(2) and cell proliferation is mediated largely through the actions of cPLA(2) whereas, sPLA(2) plays no significant role. We now also report a hitherto unsuspected contribution of iPLA(2) to this process and demonstrate that the stimulating action of EGF and IL-1beta in AA release and cell proliferation is mediated in part via a MEK and ERK-dependent pathway (but not through p38MAPK). We therefore propose that selective inhibitors of MEK and MAPK pathways may be useful in controlling AA release, eicosanoid production and cell proliferation.

L2 ANSWER 9 OF 27 MEDLINE on STN
ACCESSION NUMBER: 200106581 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10643787
TITLE: Novel strategies for opposing murine microglial activation.
AUTHOR: Paris D; Town T; Mullan M
CORPORATE SOURCE: The Roskamp Institute, University of South Florida, Tampa 33613, USA.. dparis@com1.med.usf.edu
SOURCE: Neuroscience letters, (2000 Jan 7) Vol. 278, No. 1-2, pp. 5-8.
Journal code: 7600130. ISSN: 0304-3940.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002

ENTRY DATE: Entered STN: 9 Mar 2000
 Last Updated on STN: 9 Mar 2000
 Entered Medline: 22 Feb 2000

AB Pathologic microglial activation is believed to contribute to progressive neuronal damage in neurodegenerative diseases by the release of potentially neurotoxic agents, such as pro-inflammatory cytokines including tumor necrosis factor alpha (TNF-alpha). Using cultured N9 microglial cells, we have examined the regulation of TNF-alpha following endotoxic insult with lipopolysaccharide (LPS), focusing on the role of the pro-inflammatory phospholipase A2/mitogen activated protein kinase/arachidonic acid/cyclo-oxygenase-2 cascade and the nitric oxide/cGMP pathway. Data show that various inhibitors of the PLA2 cascade markedly inhibit LPS-induced TNF-alpha release, supporting a key role of this pathway in the regulation of microglial activation. We also investigated the putative effects of cGMP-elevating agents on blocking microglial activation induced by LPS. Data show that each member of this class of cGMP-elevating compounds that we employed opposed microglial TNF-alpha release, suggesting that strengthening intracellular cGMP signaling mitigates against microglial activation. Taken together, our results suggest novel strategies for reducing microglial activation.

L2 ANSWER 10 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1999318341 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10391457
TITLE: Involvement of axonal phospholipase A2 activity in the outgrowth of adult mouse sensory axons in vitro.
AUTHOR: Hornfelt M; Ekstrom P A; Edstrom A
CORPORATE SOURCE: Department of Animal Physiology, Lund University, Sweden.
SOURCE: Neuroscience, (1999) Vol. 91, No. 4, pp. 1539-47.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 10 Sep 1999
 Last Updated on STN: 10 Sep 1999
 Entered Medline: 24 Aug 1999

AB The effect on axonal outgrowth of inhibition of phospholipase A2 activity was studied in a recently developed *in vitro* model, where dorsal root ganglia with attached spinal roots and nerve stumps from young adult mice were cultured in an extracellular matrix material (Matrigel). The phospholipase A2 inhibitors 4-bromophenacyl bromide and oleyoxyethyl phosphorylcholine dose-dependently reduced axonal outgrowth from the sciatic nerve stump. A similar inhibitory effect was seen when only the cut nerve end was exposed to the inhibitors in a compartmental culture system. The local effect of phospholipase A2 inhibition was further investigated on axons established in culture, using time-lapse recording. Exposure to phospholipase A2 inhibitors caused the retraction of filopodia extensions and a reduction in growth cone motility within a few minutes. After removal of inhibition, normal growth cone motility and axonal growth were regained. Nerve cell bodies and axons, in contrast to Schwann cells, showed immunoreactivity after staining with an antiserum against secretory phospholipase A2, and elevated levels of the enzyme could be detected after culture for 24 h. The immunoreactive protein was of approximately 170,000 molecular weight (phospholipase A2-170) as determined by sodium dodecyl sulphate-polyacrylamide gel electrophoresis and immunoblotting. The localization of phospholipase A2-170 in axons growing into the Matrigel was also demonstrated by use of a whole-mount technique. The results of this study show the importance of continuous phospholipase A2 activity for growth cone motility and axonal outgrowth in the mammalian

peripheral nerve, and suggest the involvement of an axonally localized enzyme.

L2 ANSWER 11 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1999217849 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10203186
TITLE: Role of endothelial factors in the specific response of mouse tumour-feeding arterioles to stimulation of 5-HT₁ receptors.
AUTHOR: Laemmel E; Stucker O; Vicaut E
CORPORATE SOURCE: Dept de Biophysique et INSERM U141, Hopital F. Widal, Paris, France.
SOURCE: International journal of radiation biology, (1999 Mar) Vol. 75, No. 3, pp. 365-71.
Journal code: 8809243. ISSN: 0955-3002.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 11 May 1999
Last Updated on STN: 11 May 1999
Entered Medline: 29 Apr 1999

AB PURPOSE: To investigate the possible role of endothelial mediators on the increased vasoconstriction to 5-HT₁ receptor stimulation by the host-modified arterioles feeding a Meth-A tumour implanted in the flank of female Balb/c mice. MATERIALS AND METHODS: Using intravital microscopy, the response to the topical administration of the general 5-HT₁ agonist 5-carboxamidotryptamine maleate (5-CT; 10(-6) M to 10(-4) M) by the tumour-feeding arterioles with the responses of tumour-independent arterioles and those of control arterioles from mice without tumour after antagonization or inhibition of the synthesis of endothelial mediators was compared. RESULTS: The dramatically higher vasoconstriction to 5-CT observed in tumour-feeding arterioles than in tumour-independent or control arterioles still persisted when either nitric oxide synthase, cyclooxygenase, lipoxygenase, or phospholipase A2 were inhibited or when thromboxane A2 or endothelin were antagonized. CONCLUSIONS: It was concluded that the higher reactivity to 5-HT₁ stimulation by tumour-feeding arterioles is not due to changes in endothelial mediator release but probably due to changes affecting arteriolar smooth muscle.

L2 ANSWER 12 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1998363084 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9699504
TITLE: Arachidonic acid is an autocoid mediator of the differential action of 1,25-(OH)D₃ and 24,25-(OH)D₃ on growth plate chondrocytes.
AUTHOR: Boyan B D; Sylvia V L; Curry D; Chang Z; Dean D D; Schwartz Z
CORPORATE SOURCE: Department of Orthopaedics, The University of Texas Health Science Center at San Antonio, 78284-7774, USA.. messier@uthscsa.edu
CONTRACT NUMBER: DE-05937 (United States NIDCR)
DE-08603 (United States NIDCR)
SOURCE: Journal of cellular physiology, (1998 Sep) Vol. 176, No. 3, pp. 516-24.
Journal code: 0050222. ISSN: 0021-9541.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 3 Sep 1998
Last Updated on STN: 3 Mar 2000
Entered Medline: 21 Aug 1998

AB Prior studies have shown that 24,25-(OH)2D3 and 1,25-(OH)2D3 regulate protein kinase C (PKC) in costochondral chondrocytes in a cell maturation-dependent manner, with 1,25-(OH)2D3 affecting primarily growth zone (GC) cells and 24,25-(OH)2D3 affecting primarily resting zone (RC) cells. In addition, 1,25-(OH)2D3 has been shown to increase phospholipase A2 activity in GC, while 24,25-(OH)2D3 has been shown to decrease phospholipase A2 activity in RC. Stimulation of phospholipase A2 in GC caused an increase in PKC, whereas inhibition of phospholipase A2 activity in RC cultures increased both basal and 24,25-(OH)2D3-induced PKC activity, suggesting that phospholipase A2 may play a central role in mediating the effects of the vitamin D metabolites on PKC. To test this hypothesis, RC and GC cells were cultured in the presence and absence of phospholipase A2 inhibitors (quinacrine and oleyloxyethylphosphorylcholine [OEPC]), phospholipase A2 activators (melittin and mastoparan), or arachidonic acid alone or in the presence of the target cell-specific vitamin D metabolite. PKC specific activity in the cell layer was determined as a function of time. Phospholipase A2 inhibitors decreased both basal and 1,25-(OH)2D3-induced PKC activity in GC. When phospholipase A2 activity was activated by inclusion of melittin or mastoparan in the cultures, basal PKC activity in RC was reduced, while that in GC was increased. Similarly, melittin and mastoparan decreased 24,25-(OH)2D3-induced PKC activity in RC and increased 1,25-(OH)2D3-induced PKC activity in GC. For both cell types, the addition of arachidonic acid to the culture media produced an effect on PKC activity that was similar to that observed when phospholipase A2 activators were added to the cells. These results demonstrate that vitamin D metabolite-induced changes in phospholipase A2 activity are directly related to changes in PKC activity. Similarly, exogenous arachidonic acid affects PKC in a manner consistent with activation of phospholipase A2. These effects are cell maturation- and time-dependent and metabolite-specific.

L2 ANSWER 13 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1991183640 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1901255
TITLE: Inhibitors of cytochrome P-450 attenuate the myogenic response of dog renal arcuate arteries.
AUTHOR: Kauser K; Clark J E; Masters B S; Ortiz de Montellano P R;
Ma Y H; Harder D R; Roman R J
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin,
Milwaukee 53226.
CONTRACT NUMBER: HL-29587 (United States NHLBI)
HL-33833 (United States NHLBI)
HL-36279 (United States NHLBI)
+
SOURCE: Circulation research, (1991 Apr) Vol. 68, No. 4, pp.
1154-63.
Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 26 May 1991
Last Updated on STN: 3 Feb 1997

Entered Medline: 8 May 1991

AB The role of cytochrome P-450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined. The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 microM) inhibited the greater than threefold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 microM) had no effect on this response. The cytochrome P-450 inhibitors ketoconazole (10 and 100 microM) and beta-diethyl-aminoethylidiphenylpropylacetate (SKF 525A, 10 and 100 microM) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 microM) and ketoconazole (100 microM) reduced active wall tension in renal arteries by approximately 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P-450, 1-aminobenzotriazole (75 microM) and 12-hydroxy-16-heptadecenoic acid (20 microM). The thromboxane receptor antagonist SQ 29,548 (1 or 10 microM) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 microM) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 microM) or SKF 525A (100 microM). Microsomes (1 mg/ml) prepared from small renal arteries (200-500 microns) and incubated with [1-14C]arachidonic acid (0.5 mu Ci, 50 microM) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase high-performance liquid chromatography. The formation of this product was inhibited by both ketoconazole and SKF 525A at concentrations of 10 and 100 microM. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P-450 metabolites of endogenous fatty acids in the myogenic response.

L2 ANSWER 14 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:829238 CAPLUS

DOCUMENT NUMBER: 141:329077

TITLE: Interactions of 12-lipoxygenase with phospholipase A2 isoforms following platelet activation through the glycoprotein VI collagen receptor

AUTHOR(S): Coffey, Marcus J.; Coles, Barbara; Locke, Matthew; Bermudez-Fajardo, Alexandra; Williams, P. Claire; Jarvis, Gavin E.; O'Donnell, Valerie B.

CORPORATE SOURCE: Department of Medical Biochemistry and Immunology, Wales College of Medicine, Cardiff University, Cardiff, CF14 4XN, UK

SOURCE: FEBS Lett. (2004), 576(1-2), 165-168
CODEN: FEBBL; ISSN: 0014-5793

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Recent studies implicate the collagen receptor, glycoprotein VI (GPVI) in activation of platelet 12-lipoxygenase (p12-LOX). Herein, we show that GPVI-stimulated 12-hydroperoxyeicosatetraenoic acid (H(P)ETE) synthesis is inhibited by palmitoyl trifluoromethyl ketone or oleyloxyethylphosphocholine, but not bromoenol lactone, implicating secretory and cytosolic, but not calcium-independent phospholipase A2 (PLA2) isoforms. Also, following GPVI activation, 12-LOX co-immunoprecipitates with both cytosolic and secretory PLA2 (sPLA2). Finally, venoms containing sPLA2 acutely activate p12-LOX in a dose-dependent manner. This study shows that platelet 12-H(P)ETE generation utilizes arachidonate substrate from both c- and sPLA2 and that 12-LOX functionally associates with both PLA2 isoforms.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 15 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:748810 CAPLUS
DOCUMENT NUMBER: 142:129558
TITLE: Phospholipase A2 in salivary glands isolated from tobacco hornworms, *Manduca sexta*
AUTHOR(S): Tunaz, Hasan; Stanley, David W.
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska-Lincoln, Lincoln, NE, 68583-0816, USA
SOURCE: Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2004), 139B(1), 27-33
CODEN: CBPBB8; ISSN: 1096-4959
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A phospholipase A2 (PLA2) associated with the salivary glands of *M. sexta* is described. This enzyme was able to hydrolyze arachidonic acid from the sn-2 position of phospholipases. The addition of the Ca²⁺-chelator, EGTA, or Ca²⁺, to the Tris reaction buffer impaired the PLA2 activity, from which it was inferred that the enzyme required very low concns. of Ca²⁺ or perhaps other ions for optimal activity. PLA2 activity was sensitive to protein concentration (highest activity at 25 µg protein/mL), reaction time (optimal at 30 min), buffer pH (optimal at pH 8-10), and reaction temperature (optimal range 18-38°). The salivary gland PLA2 was sensitive to the site-specific inhibitor, oleyloxyethylphosphorylcholine and stable to freezing at -80°, but not -20°. The biol. significance of this enzyme may relate to hydrolysis of fatty acid moieties from dietary phospholipases for absorption by midgut epithelia. This salivary gland enzyme may also be responsible for killing food-borne bacteria.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 16 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:447810 CAPLUS
DOCUMENT NUMBER: 139:286724
TITLE: 1 α ,25(OH)2D3 causes a rapid increase in phosphatidylinositol-specific PLC- β activity via phospholipase A2-dependent production of lysophospholipid
AUTHOR(S): Schwartz, Z.; Shaked, D.; Hardin, R. R.; Gruwell, S.; Dean, D. D.; Sylvia, V. L.; Boyan, B. D.
CORPORATE SOURCE: Department of Biomedical Engineering, Georgia Institute of Technology, Atlanta, GA, 30332, USA
SOURCE: Steroids (2003), 68(5), 423-437
CODEN: STEDAM; ISSN: 0039-128X
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 1 α ,25(OH)2D3 activates protein kinase C (PKC) in rat growth plate chondrocytes via mechanisms involving phosphatidylinositol-specific phospholipase C (PI-PLC) and phospholipase A2 (PLA2). The purpose of this study was to determine if 1 α ,25(OH)2D3 activates PI-PLC directly or through a PLA2-dependent mechanism. We determined which PLC isoforms are present in the growth plate chondrocytes, and determined which isoform(s) of PLC is(are) regulated by 1 α ,25(OH)2D3. Inhibitors and activators of PLA2 were used to assess the inter-relationship between these two phospholipid-signaling pathways. PI-PLC activity in lysates of pre-hypertrophic and upper hypertrophic zone (growth zone) cells that were incubated with 1 α ,25(OH)2D3, was increased within 30 s with peak activity at 1-3 min. PI-PLC activity in resting zone cells was unaffected

by 1 α ,25(OH)2D3. 1 β ,25(OH)2D3, 24R,25(OH)2D3, actinomycin D and cycloheximide had no effect on PLC in lysates of growth zone cells. Thus, 1 α ,25(OH)2D3 regulation of PI-PLC enzyme activity is stereospecific, cell maturation-dependent, and nongenomic. PLA2-activation (mastioparan or melittin) increased PI-PLC activity to the same extent as 1 α ,25(OH)2D3; PLA2-inhibition (quinacrine, oleyloxyethylphosphorylcholine (OEPC), or AACOCF3) reduced the effect of 1 α ,25(OH)2D3. Neither arachidonic acid (AA) nor its metabolites affected PI-PLC. In contrast, lysophosphatidylcholine (LPC) and lysophosphatidylethanolamine (LPE) activated PI-PLC (LPE:LPC). 1 α ,25(OH)2D3 stimulated PI-PLC and PKC activities via Gq; GDP β S inhibited activity, but pertussis toxin did not. RT-PCR showed that the cells express PLC- β 1a, PLC- β 1b, PLC- β 3 and PLC- γ 1 mRNA. Antibodies to PLC- β 1 and PLC- β 3 blocked the 1 α ,25(OH)2D3 effect; antibodies to PLC- δ and PLC- γ did not. Thus, 1 α ,25(OH)2D3 regulates PLC- β through PLA2-dependent production of lysophospholipid.

REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 17 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:112769 CAPLUS

DOCUMENT NUMBER: 139:346941

TITLE: Enzymatic activity and inhibition of the neurotoxic complex vipoxin from the venom of Vipera ammodytes meridionalis

AUTHOR(S): Noetzel, Corinna; Chandra, Vikas; Perbandt, Markus; Rajashankar, Kanagalaghatta; Singh, Tej; Aleksiev, Boris; Kalkura, Narayana; Genov, Nicolay; Betzel, Christian

CORPORATE SOURCE: Institute of Medical Biochemistry and Molecular Biology, University Hospital Eppendorf, Hamburg, 22603, Germany

SOURCE: Zeitschrift fuer Naturforschung, C: Journal of Biosciences (2002), 57(11/12), 1078-1083

CODEN: ZNCBDA; ISSN: 0939-5075

PUBLISHER: Verlag der Zeitschrift fuer Naturforschung
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vipoxin from the venom of Vipera ammodytes meridionalis is an unique neurotoxic complex between a toxic phospholipase A2 and a highly homologous non-toxic protein inhibitor. It is an example of evolution of a catalytic and toxic function into inhibitory and non-toxic one. The activity of the V. ammodytes meridionalis toxin is 1.7 times higher than that of the closely related (92% sequence identity) neurotoxic complex RV4/RV7 from the venom of Vipera russelli formosensis. The enhanced enzymic activity of vipoxin is attributed to limited structural changes, in particular to the substitutions G54R and Q78K in the PLA2 subunit of the complex and to the T84R substitution in the inhibitor.

Oleyloxyethylphosphocholine, aristolochic acid and vitamin E suppressed the enzymic activity of vipoxin and its isolated PLA2 subunit. These compds. influence inflammatory processes in which PLA2 is implicated. The peptide Lys-Ala-Ile-Tyr-Ser, which is an integral part of the PLA2 components of the two neurotoxic complexes from V. ammodytes meridionalis and V. russelli formosensis (sequence 70-74) activated vipoxin increasing its PLA2 activity by 23%. This is in contrast to the inhibitory effect of the resp. pentapeptides with 70-74 sequences on other group II PLA2s. Surprisingly, the same peptide inhibited 46% of the V. russelli formosensis PLA2 activity. The limited changes in the structure of the two highly homologous neurotoxins lead to considerable differences in their interaction with native peptides.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 18 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
 ACCESSION NUMBER: 2000:735786 CAPLUS
 DOCUMENT NUMBER: 133:345041
 TITLE: Investigation into the involvement of phospholipases A2 and MAP kinases in modulation of AA release and cell growth in A549 cells
 AUTHOR(S): Choudhury, Qamrul G.; McKay, Diane T.; Flower, Roderick J.; Croxtall, Jamie D.
 CORPORATE SOURCE: Department of Biochemical Pharmacology, The William Harvey Research Institute, St. Bartholomew's and the Royal London School of Medicine and Dentistry (Queen Mary and Westfield College, London, EC1M 6BQ, UK
 SOURCE: British Journal of Pharmacology (2000), 131(2), 255-265
 CODEN: BJPCBM; ISSN: 0007-1188
 PUBLISHER: Nature Publishing Group
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The authors have investigated the contribution of specific PLA2s to eicosanoid release from A549 cells by using specific inhibitors of secretory PLA2 (ONO-RS-82 and oleyloxyethylphosphocholine), cytosolic PLA2 (AACOCF3 and MAFP) and calcium-independent PLA2 (HELSS, MAFP and PACOCF3). Similarly, by using specific inhibitors of p38 MAPK (SB 203580), ERK1/2 MAPK (Apigenin) and MEK1/2 (PD 98059) the authors have further evaluated potential pathways of AA release in this cell line. ONO-RS-82 and oleyloxyethylphosphocholine had no significant effect on EGF or IL-1 β stimulated 3H-AA or PGE2 release or cell proliferation. AACOCF3, HELSS, MAFP and PACOCF3 significantly inhibited both EGF and IL-1 β stimulated 3H-AA and PGE2 release as well as cell proliferation. Apigenin and PD 98059 significantly inhibited both EGF and IL-1 β stimulated 3H-AA and PGE2 release and cell proliferation, whereas, SB 203580 had no significant effect on EGF or IL-1 β stimulated 3H-AA release, or cell proliferation but significantly suppressed EGF or IL-1 β stimulated PGE2 release. These results confirm that the liberation of AA release, generation of PGE2 and cell proliferation is mediated largely through the actions of cPLA2 whereas, sPLA2 plays no significant role. The authors now also report a hitherto unsuspected contribution of iPLA2 to this process and demonstrate that the stimulating action of EGF and IL-1 β in AA release and cell proliferation is mediated in part via a MEK and ERK-dependent pathway (but not through p38MAPK). The authors therefore propose that selective inhibitors of MEK and MAPK pathways may be useful in controlling AA release, eicosanoid production and cell proliferation.
 REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
 ACCESSION NUMBER: 1999:641140 CAPLUS
 DOCUMENT NUMBER: 132:21206
 TITLE: Tissue polyunsaturated fatty acids and a digestive phospholipase A2 in the primary screwworm, *Cochliomyia hominivorax*
 AUTHOR(S): Nor Aliza, A. R.; Rana, R. L.; Skoda, S. R.; Berkebile, D. R.; Stanley, D. W.
 CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska, Lincoln, NE, USA
 SOURCE: Insect Biochemistry and Molecular Biology (1999), 29(11), 1029-1038
 CODEN: IBMBES; ISSN: 0965-1748
 PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB The authors report on the presence of arachidonic acid in larval and adult tissues of the primary screwworm, *Cochliomyia hominivorax*, and of the secondary screwworm, *C. macellaris*. Arachidonic acid is present in the phospholipids of whole animal exts. of both species. This fatty acid appears to be accumulated during the larval stages, because proportions of arachidonic acid were higher in adults than in larvae. These insects probably obtain the arachidonic acid from dietary phospholipids. The authors also report on a phospholipase A2 activity in midgut preps. from third instars of the primary screwworm. Phospholipase A2 is responsible for hydrolyzing fatty acids from the sn-2 position of dietary phospholipids to release essential fatty acids. The screwworm enzyme is similar to mammalian digestive phospholipase A2 because it depends on calcium for high catalytic activity, it is sensitive to the site-specific inhibitor oleyloxyethylphosphorylcholine, and it interacts with heparin. The authors further characterized the screwworm midgut phospholipase A2 by altering the reaction conditions, including reaction time, radioactive substrate concentration, protein concentration, pH and temperature. The authors speculate that the biol. significance of this enzyme relates to acquiring essential fatty acids, including arachidonic acid, from dietary phospholipids.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 20 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1998:626204 CAPLUS

DOCUMENT NUMBER: 129:328495

ORIGINAL REFERENCE NO.: 129:66942h,66943a

TITLE: A digestive phospholipase A2 in larval mosquitoes,
Aedes aegypti

AUTHOR(S): Aliza, A. R. Nor; Stanley, David W.

CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, Department
of Entomology, University of Nebraska, Lincoln, NE,
68583-0816, USA

SOURCE: Insect Biochemistry and Molecular Biology (1998),
28(8), 561-569

CODEN: IBMBES; ISSN: 0965-1748

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We report on the presence of phospholipase A2 activity in whole larva and midguts of the yellow fever mosquito *A. aegypti*. Phospholipase A2 is responsible for hydrolyzing fatty acids from the sn-2 position of dietary phospholipids to release essential fatty acids for normal larval and adult growth. In contrast to the mammalian digestive phospholipase A2 background, the *A. aegypti* phospholipase A2 was Ca²⁺ independent. We further characterized the mosquito midgut phospholipase A2 by altering the reaction conditions including incubation time, protein concns., pH, and temperature. The site-specific PLA2 inhibitor oleyloxyethylphosphorylcholine failed to inhibit the enzyme at concns. <5000 μM. The phospholipase A2 activity was consistently high throughout the 4th instar, but fell to very low levels on the 1st day of pupation which is a non-feeding stage. The enzyme is regulated with respect to feeding activity because fasting and re-feeding modified the larval digestive PLA2 activity.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 21 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1998:503604 CAPLUS

DOCUMENT NUMBER: 129:226089
ORIGINAL REFERENCE NO.: 129:45841a, 45844a
TITLE: Arachidonic acid is an autocoid mediator of the differential action of 1,25-(OH)2D3 and 24,25-(OH)2D3 on growth plate chondrocytes
AUTHOR(S): Boyan, B. D.; Sylvia, V. L.; Curry, D.; Chang, Z.;
Dean, D. D.; Schwartz, Z.
CORPORATE SOURCE: Department of Orthopaedics, The University of Texas Health Science Center at San Antonio, San Antonio, TX, 78284-7774, USA
SOURCE: Journal of Cellular Physiology (1998), 176(3), 516-524
CODEN: JCLLAX; ISSN: 0021-9541
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Prior studies have shown that 24,25-(OH)2D3 and 1,25-(OH)2D3 regulate protein kinase C (PKC) in costochondral chondrocytes in a cell maturation-dependent manner, with 1,25-(OH)2D3 affecting primarily growth zone (GC) cells and 24,25-(OH)2D3 affecting primarily resting zone (RC) cells. In addition, 1,25-(OH)2D3 has been shown to increase phospholipase A2 activity in GC, while 24,25-(OH)2D3 has been shown to decrease phospholipase A2 activity in RC. Stimulation of phospholipase A2 in GC caused an increase in PKC, whereas inhibition of phospholipase A2 activity in RC cultures increased both basal and 24,25-(OH)2D3-induced PKC activity, suggesting that phospholipase A2 may play a central role in mediating the effects of the vitamin D metabolites on PKC. To test this hypothesis, RC and GC cells were cultured in the presence and absence of phospholipase A2 inhibitors (quinacrine and oleyloxyethylphosphorylcholine [OEPC]), phospholipase A2 activators (melittin and mastoparan), or arachidonic acid alone or in the presence of the target cell-specific vitamin D metabolite. PKC specific activity in the cell layer was determined as a function of time. Phospholipase A2 inhibitors decreased both basal and 1,25-(OH)2D3-induced PKC activity in GC. When phospholipase A2 activity was activated by inclusion of melittin or mastoparan in the cultures, basal PKC activity in RC was reduced, while that in GC was increased. Similarly, melittin and mastoparan decreased 24,25-(OH)2D3-induced PKC activity in RC and increased 1,25-(OH)2D3-induced PKC activity in GC. For both cell types, the addition of arachidonic acid to the culture media produced an effect on PKC activity that was similar to that observed when phospholipase A2 activators were added to the cells. These results demonstrate that vitamin D metabolite-induced changes in phospholipase A2 activity are directly related to changes in PKC activity. Similarly, exogenous arachidonic acid affects PKC in a manner consistent with activation of phospholipase A2. These effects are cell maturation- and time-dependent and metabolite-specific.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 22 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:481432 CAPLUS
DOCUMENT NUMBER: 129:186925
ORIGINAL REFERENCE NO.: 129:37913a, 37916a
TITLE: A digestive phospholipase A2 in midguts of tobacco hornworms, *Manduca sexta* L.
AUTHOR(S): Rana, Rico L.; Sarah, Gautam; Stanley, David W.
CORPORATE SOURCE: Insect Biochemistry/Physiology Laboratory, Department of Entomology, University of Nebraska, Lincoln, NE, 68583-0816, USA
SOURCE: Journal of Insect Physiology (1998), 44(3-4), 297-303
CODEN: JIPHAF; ISSN: 0022-1910
PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB We hypothesized that phospholipase A2 (PLA2) is a common feature of insect digestive physiol. PLA2 hydrolyzes polyunsatd. fatty acids (PUFAs) associated with the sn-2 position of phospholipids (PLs). We describe here a PLA2 from midgut contents of the tobacco hornworm, *M. sexta*. The enzyme is sensitive to pH (inactivated at low pH), protein concentration (<1.6 µg/µl), substrate concentration (<1.4 nmol/reaction), temperature (<30°), and incubation time. PLA2 activity is higher in fed than in starved larvae, and enzyme activity is associated with the midgut contents, rather than the midgut epithelium of fed larvae. All known secretory PLA2s, except for a PLA2 in venom of the marine snail, *Conus magus*, require high calcium concns. for catalysis, but the Manduca PLA2 appears to be calcium independent, and it exhibits increased PLA2 activity in the presence of a calcium chelator, EGTA. In addition, the partially purified Manduca PLA2 is not inhibited by the phospholipid analog, oleyloxyethylphosphorylcholine. These findings suggest that the Manduca digestive PLA2 may represent another novel form of PLA2.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 23 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1993:188162 CAPLUS

DOCUMENT NUMBER: 118:188162

ORIGINAL REFERENCE NO.: 118:32247a,32250a

TITLE: The possible involvement of protein kinase C and phospholipase A2 in Hydra tentacle regeneration

AUTHOR(S): De Petrocellis, L.; Di Marzo, V.; Cinino, G.

CORPORATE SOURCE: Ist. Cibern., CNR, Arco Felice, I-80072, Italy

SOURCE: Experientia (1993), 49(1), 57-64

CODEN: EXPEAM; ISSN: 0014-4754

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The participation of protein kinase C (PKC) in the regeneration of tentacles of *Hydra vulgaris* was studied. Regeneration was induced by 1,2-sn-di octanoyl-glycerol (dic8) and the novel diterpenoid diacylglycerol verrucosin B (VB), a potent PKC activator extracted from marine sources. VB substantially increased Hydra average tentacle number (ATN) at concns. 10,000 times lower than those needed for dic8 to exert an analogous effect. When both synthetic and natural VB analogs were tested, the structure/activity relation found in Hydra tentacle regeneration was identical to that known for DAG-induced activation of PKC in vitro. VB-induced increases in ATN was strongly counteracted by the PKC inhibitors sphingosine and A3, but was not synergistic with a 10-fold increase of extracellular Ca²⁺ concentration or with an increase of intracellular Ca²⁺ concentration obtained either with the

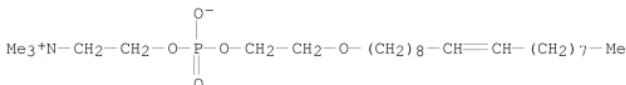
ionophore A 23187 or with thapsigargin. This suggested the involvement of a non-Ca²⁺-dependent PKC in VB-triggered Hydra tentacle regeneration. The involvement of phospholipase A2 (PLA2) activation in Hydra regenerative processes was studied using the novel site-specific inhibitor of the enzyme, oleyloxyethylphosphorylcholine (OOPC), which brought about a striking inhibition of ATN in the low micromolar range. This effect was reversed by arachidonic acid (AA), while an enhancement of ATN was also observed with an inhibitor of AA uptake from membrane phospholipids, thus suggesting that PLA2-catalyzed liberation of AA is involved in Hydra tentacle regeneration. OOPC also blocked verrucosin B-induced PKC-mediated enhancement of ATN, thus suggesting that this effect is also mediated by PLA2 activation. ATN was increased also by compound 48/80, a direct activator of pertussis toxin-sensitive GTP-binding proteins, and this effect was counteracted by pertussis toxin pretreatment. None of the known AA cascade inhibitors exhibited an effect on ATN comparable to that exerted by OOPC, but, surprisingly, the cyclooxygenase inhibitor

indomethacin strongly enhanced ATN, thus suggesting that prostanoids might effect a neg. control on Hydra regenerative processes. This represents the 1st attempt to study the implication of >1 biochem. pathway as a signaling event in the hydroid regenerative processes.

L2 ANSWER 24 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:401629 CAPLUS
DOCUMENT NUMBER: 115:1629
ORIGINAL REFERENCE NO.: 115:342h,343a
TITLE: Inhibitors of cytochrome P-450 attenuate the myogenic response of dog renal arcuate arteries
AUTHOR(S): Kausler, Katalin; Clark, Joan E.; Masters, Bettie Sue; Ortiz de Montellano, Paul R.; Ma, Yunn Hwa; Harder, David R.; Roman, Richard J.
CORPORATE SOURCE: Dep. Physiol., Med. Coll. Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Circulation Research (1991), 68(4), 1154-63
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The role of cytochrome P 450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined. The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 μ M) inhibited the 3-fold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 μ M) had no effect on this response. The cytochrome P 450 inhibitors ketoconazole (10 and 100 μ M) and β -diethyl-aminoethylidiphenylpropylacetate (SKF 525A, 10 and 100 μ M) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 μ M) and ketoconazole (100 μ M) reduced active wall tension in renal arteries by approx. 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P 450, 1-aminobenzotriazole (75 μ M) and 12-hydroxy-16-heptadecenoic acid (20 μ M). The thromboxane receptor antagonist SQ 29,548 (1 of 10 μ M) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 μ M) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 μ M) or SKF 525A (100 μ M). Microsomes (1 mg/mL) prepared from small renal arteries (200-500 μ m) and incubated with [l-14C]arachidonic acid (0.5 μ Ci, 50 μ M) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase HPLC. The formation of this product was inhibited by both ketoconazole and SKF 525A at concns. of 10 and 100 μ M. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P 450 metabolites of endogenous fatty acids in the myogenic response.

L2 ANSWER 25 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:406131 CAPLUS
DOCUMENT NUMBER: 103:6131
ORIGINAL REFERENCE NO.: 103:1103a,1106a
TITLE: A new efficient and versatile synthesis of alkyl phosphorylcholines
AUTHOR(S): Magolda, R. L.; Johnson, P. R.
CORPORATE SOURCE: Cent. Res. Dev. Dep., E. I. du Pont de Nemours and Co., Wilmington, DE, 19898, USA
SOURCE: Tetrahedron Letters (1985), 26(9), 1167-70
DOCUMENT TYPE: Journal
LANGUAGE: English

OTHER SOURCE(S): CASREACT 103:6131
 AB Phosphorylcholines ROP(O)(O-)OCH₂CH₂N+Me₃ [R = Me(CH₂)_n, Me(CH₂)₇CH(CH₂)₈, Me(CH₂)mS(CH₂)₃, Me(CH₂)₇CH:CH(CH₂)₈S(CH₂)₃, Me(CH₂)mOCH₂CH₂, Me(CH₂)₇CH:CH(CH₂)₈OCH₂CH₂; m = 15,17; n = 5,7,11,17] were prepared in 35-50% overall yield by treating ROH with POCl₃, followed by ethylene glycol and treating the resulting cyclic phosphates with Me₃N.
 IT 96720-06-8P
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (preparation of)
 RN 96720-06-8 CAPLUS
 CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-, inner salt 4-oxide (CA INDEX NAME)



L2 ANSWER 26 OF 27 WPIDS COPYRIGHT 2008 THOMSON REUTERS on STN
 ACCESSION NUMBER: 2000-171073 [15] WPIDS
 DOC. NO. CPI: C2000-053194 [15]
 TITLE: Modifying vasoactivity and inflammatory actions in microglia and neurons comprises regulating a soluble alphabeta pro-inflammatory pathway, useful for treating vascular diseases, e.g. cerebral amyloid angiopathy and vascular amyloidosis
 DERWENT CLASS: B04
 INVENTOR: MULLAN M J; PARIS D; TOWN T C
 PATENT ASSIGNEE: (UYSF-N) UNIV SOUTH FLORIDA
 COUNTRY COUNT: 84

PATENT INFO ABBR.:

| PATENT NO | KIND | DATE | WEEK | LA | PG | MAIN IPC |
|---------------|------|----------|-----------|----|--------|----------|
| WO 2000002561 | A1 | 20000120 | (200015)* | EN | 77[29] | |
| AU 9949948 | A | 20000201 | (200028) | EN | | |

APPLICATION DETAILS:

| PATENT NO | KIND | APPLICATION | DATE |
|---------------|------|-----------------|----------|
| WO 2000002561 | A1 | WO 1999-US15947 | 19990713 |
| AU 9949948 | A | AU 1999-49948 | 19990713 |

FILING DETAILS:

| PATENT NO | KIND | PATENT NO |
|--------------|----------|-----------------|
| AU 9949948 A | Based on | WO 2000002561 A |

PRIORITY APPLN. INFO: US 1998-92570P 19980713
 AN 2000-171073 [15] WPIDS
 AB WO 2000002561 A1 UPAB: 20060116
 NOVELTY - Modifying vasoactivity comprises regulating a soluble alphabeta pro-inflammatory pathway.
 DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
 (1) a pharmaceutical composition comprising an effective amount of

a soluble alphabeta pro-inflammatory pathway regulator and a carrier;
(2) a diagnostic method including detecting modification of the soluble alphabeta pro-inflammatory pathway; and
(3) a method of modifying inflammatory actions in microglia and neurons by regulating a soluble alphabeta pro-inflammatory pathway.

ACTIVITY - Vasoactive.

MECHANISM OF ACTION - Signal transduction pathway (e.g. sPLA2/MAPK/cPLA2/AA/LOX/COX) modulators.

In tests to evaluate the effect of alphabeta on microglial LTB4 release, soluble alphabeta treatment of the murine microglial cell line, N9, resulted in an increased release of LTB4. COX-2 inhibition (via the COX-2 specific inhibitor, NS-398, 50 microM) resulted in complete blockade of LTB4 release. Furthermore, inhibition of p38 MAPK by the specific inhibitor SB202190 (5 microM) or inhibition of MEK1/2 via PD98059 (25 microM) each resulted in complete blockade of alphabeta-induced microglial LTB4 release.

USE - The method is for treating patients with vascular disease by modifying an intracellular soluble alphabeta pro-inflammatory pathway (claimed). Vascular diseases to be treated include cerebral amyloid angiopathy and vascular amyloidosis.

ADVANTAGE - The methods reduce neuronal cell death associated with the pro-inflammatory pathway and vasoactivity.

L2 ANSWER 27 OF 27 USPATFULL on STN

ACCESSION NUMBER: 2006127387 USPATFULL

TITLE: Coupling factor 6 inhibitor and potentiator and use thereof

INVENTOR(S): Osanai, Tomohiro, Nakatsugaru-gun, JAPAN

Magota, Koji, Takatsuki, JAPAN

PATENT ASSIGNEE(S): Daiichi Suntory Pharma Co.,Ltd., Tokyo, JAPAN (non-U.S. corporation)

| | NUMBER | KIND | DATE |
|---------------------|----------------|------|-----------------------|
| PATENT INFORMATION: | US 7049079 | B1 | 20060523 |
| | WO 2001021205 | | 20010329 |
| APPLICATION INFO.: | US 2000-831951 | | 20000803 (9) |
| | WO 2000-JP5210 | | 20000803 |
| | | | 20010814 PCT 371 date |

| | NUMBER | DATE |
|-----------------------|----------------|----------|
| PRIORITY INFORMATION: | JP 1999-264687 | 19990917 |

DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED

PRIMARY EXAMINER: Nolan, Patrick J.

LEGAL REPRESENTATIVE: White, Jr., Paul E., Manelli Denison & Selter PLLC

NUMBER OF CLAIMS: 11

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 25 Drawing Figure(s); 25 Drawing Page(s)

LINE COUNT: 1391

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods of measuring the presence/absence of a coupling factor 6, which is a subunit of H⁺-transporting ATP synthase-H⁺-ATP synthase present in the mitochondrial inner membrane, in the blood and the concentration thereof are provided. Further, relations among the coupling factor 6 level in the blood and diseases and relations among the inhibition of the effect of the coupling factor and therapeutic effects on diseases are clarified and thus techniques for diagnosing and treating these diseases are provided.

The present invention provides a vector containing a DNA encoding the

coupling factor 6 or fragment thereof; a transformant transformed by this vector; and a method of producing the coupling factor 6 and its fragment. The present invention further provides an antibody reacting specifically with the coupling factor 6; a process of producing the antibody; and a method of assaying the coupling factor 6.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 11:21:27 ON 30 OCT 2008)

FILE 'REGISTRY' ENTERED AT 11:21:51 ON 30 OCT 2008
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYL"/CN 25

L1 1 S 96720-06-8/RN

FILE 'MEDLINE, CAPLUS, WPIDS, USPATFULL' ENTERED AT 11:24:12 ON 30 OCT 2008

L2 27 S L1 OR OLEYLOXYETHYLPHOSPHORYLCHOLINE OR OLEYLOXYETHYLPHOSPHOC

=> s 12 and alzheimer

L3 0 L2 AND ALZHEIMER

=> s 12 and alzheimer's

MISMATCHED QUOTE 'ALZHEIMER'S'

Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s 12 and vasoactivity

L4 1 L2 AND VASOACTIVITY

=> d 14

L4 ANSWER 1 OF 1 WPIDS COPYRIGHT 2008 THOMSON REUTERS on STN
AN 2000-171073 [15] WPIDS
DNC C2000-053194 [15]
TI Modifying vasoactivity and inflammatory actions in microglia and neurons comprises regulating a soluble alphabeta pro-inflammatory pathway, useful for treating vascular diseases, e.g. cerebral amyloid angiopathy and vascular amyloidosis
DC B04
IN MULLAN M J; PARIS D; TOWN T C
PA (UYSF-N) UNIV SOUTH FLORIDA
CYC 84
PI WO 2000002561 A1 20000120 (200015)* EN 77[29]
AU 9949948 A 20000201 (200028) EN
ADT WO 2000002561 A1 WO 1999-US15947 19990713; AU 9949948 A AU 1999-49948 19990713
FDT AU 9949948 A Based on WO 2000002561 A
PRAI US 1998-92570P 19980713
IPCR A61K0031-00 [I,A]; A61K0031-00 [I,C]; A61K0031-121 [I,A]; A61K0031-121 [I,C]; A61K0031-165 [I,A]; A61K0031-165 [I,C]; A61K0031-18 [I,A]; A61K0031-18 [I,C]; A61K0031-21 [I,C]; A61K0031-27 [I,A]; A61K0031-352 [I,A]; A61K0031-352 [I,C]; A61K0031-357 [I,C]; A61K0031-36 [I,A]; A61K0031-366 [I,C]; A61K0031-403 [I,C]; A61K0031-4045 [I,A]; A61K0031-405 [I,A]; A61K0031-415 [I,A]; A61K0031-415 [I,C]; A61K0031-4427 [I,C]; A61K0031-4439 [I,A]

=> d his

(FILE 'HOME' ENTERED AT 11:21:27 ON 30 OCT 2008)

FILE 'REGISTRY' ENTERED AT 11:21:51 ON 30 OCT 2008
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYL"/CN 25

L1 1 S 96720-06-8/RN

FILE 'MEDLINE, CAPLUS, WPIDS, USPATFULL' ENTERED AT 11:24:12 ON 30 OCT
2008

L2 27 S L1 OR OLEYLOXYETHYLPHOSPHORYLCHOLINE OR OLEYLOXYETHYLPHOSPHOC
L3 0 S L2 AND ALZHEIMER
L4 1 S L2 AND VASOACTIVITY

=>

---Logging off of STN---

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Executing the logoff script...

=> LOG Y

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|--|------------------|---------------|
| FULL ESTIMATED COST | 80.94 | 84.99 |
| DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION |
| CA SUBSCRIBER PRICE | -9.60 | -9.60 |

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